VOLUME - 12, ISSUE - 04, APRIL - 2023 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Periodontology

PLASMA CELL GINGIVITIS- CASE REPORT

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ABSTRACT Diagnosis is not the end but the beginning of clinical practice (Martin Fischer). Plasma cell gingivitis is a rare condition of undetermined origin characterized by diffuse and massive infiltration of plasma cells into the subepithelial connective tissue. This atypical gingivostomatitis is generally associated with chewing gum, chilly, pepper, environmental, food flavoring allergens, use of mint, candy and toothpaste ingredients. Though the exact etiology is not known allergic reaction seems to be a plausible explanation. Plasma cell gingivitis was first reported in the United States and it is a rare condition, the course of which is not fully known. We discuss a case of plasma cell gingivitis which has been diagnosed on histopathological basis and treated by non- surgical procedure. This paper presents its etiology clinical features and highlights the treatment modalities.

KEYWORDS : Plasma cells, antigen, hypersensitivity, exocytosis, micro abscess.

INTRODUCTION:

Esthetics is a science of beauty, which is the particular detail of an animate object that makes it appealing to the eye. Gingival health and appearance are the essential components of an attractive smile. Gingivitis is the inflammation of the gingiva. Plasma cells are normally found in the bone marrow and intestinal tract and plasma cell infiltrate is found in the connective tissue can be seen in autoimmune, reactive and idiopathic conditions. Anatomical site with plasma cell infiltration in connective tissue are vulva [1], gingiva, buccal mucosa, palate, nasal aperture, lips, tongue, epiglottis, larynx, pharynx, conjunctiva, skin and other intact mucosa [2]. Plasma cell gingivitis is the term used for the diseases involving the gingiva with inflammation of gingiva resulting from a hypersensitivity reaction. Also referred as atypical gingivitis, plasma cell gingivostomatitis which consists of mild marginal gingival enlargement that extends to attached gingiva. Plasma cell gingivitis was first reported in united states and is also referred to as atypical gingivitis, plasma cell gingivostomatitis, allergic gingivostomatitis, unusual gingivostomatitis and idiopathic gingivostomatitis, consist of mild marginal gingival enlargement that extends to the attached gingiva and it is a rare benign condition, the course of which is not fully known. Patterns of gingival alteration was brought to attention during late 1960s and early 1970s.

HISTORY:

Kerr et al reported a case of plasma cell gingivitis in 1971 and the cause stated was an allergic reaction to the constituents of a chewing gum. Other etiological factors stated in the development of plasma cell gingivitis are flavoring agents such as cinnamon aldehyde [3] cinnamon in chewing gums and dentifrices. After the antigen exposure some Bcells differentiate to form plasma cells dedicated to the production and secretion of antibodies of the IgM isotypes. Memory Bcells give rise to plasma cells on secondary exposure to antigen and produce high – affinity antibodies of the appropriate isotype [4]. Cessation of the exposure to allergens brings resolution of the lesion [5].

Clinical Features:

- Plasma cell gingivitis is more prevalent in young women.
- Initial symptoms are in the mouth, which often is intensified by dentifrices and hot or spicy food.
- Mild or moderate marginal gingival enlargement extends up to the attached gingiva and in severe cases extends to the buccal and vestibular mucosa [6].
- The entire free and attached gingiva demonstrate a diffuse enlargement with bright erythema and loss of stippling.
- Erythematous and friable and sometimes granular with loss of stippling and bleeds easily and does not induce a loss of attachment [7].
- Very rarely Plasma cell gingivitis also shows a tongue involvement resulting in erythematous enlargement with tumors, mild ulceration and loss of the typical dorsal coating.

Histopathology:

Baskar et al first gave a detailed description of the lesion in the gingiva, complete with histopathological examination [8]. Intense hyperemic edema and inflammation of the gingiva resulting from a hypersensitivity reaction. A dense plasma cell infiltrate is seen in lamina propria. Epithelium shows psoriasiform hyperplasia, spongiosis and inflammatory cell infiltration with intense exocytosis and neutrophilic micro abscesses [9]. Ultrastructurally there is a damage in lower spinous and basal cell layers. The underlying connective tissue contains a dense infiltrate of plasma cells [10] that also extends to the oral epithelium, inducing a dissecting type of injury [11]. A foci of plasma cells separated by collagen septae present in the connective tissue with no cellular atypia present.

Case Report:

A 22-year-old female patient reported to the department of periodontics with the chief complaint of redness and swollen gums in the upper and lower anterior region for past 8 months which was gradual in onset and increased with time with poor esthetics. Patient gave history of change in the tooth paste before 8 months. Patient had proper oral hygiene habits without any deleterious effects. The family and medical history of the patient was insignificant.

Clinical Examination

Gingival examination revealed generalized erythematous marginal gingiva and attached gingiva generalized loss of scalloping with rolled out contour of the gingiva and blunt

interdental papilla. The consistency of the gingiva was soft and edematous. Clinically the patient appeared to have severe inflammation of the gums both maxillary and mandibular with bleeding on provocation. The probing depth was recorded as 5mm around the involved teeth in maxillary anteriors and 4-5mm in mandibular teeth. On digital pressure exudate seems to be nil. The position of gingiva was above the CEJ in 13-23,33-43 and bleeding on probing was present. Hard tissue examination revealed total of 28 teeth and decay in 18,17,26,27,37 with angles class I molar relationship. Dental fluorosis was present with impacted 38,48.

Based on the clinical findings this condition may be allergic variant due to the use of herbal tooth paste.Further investigations were carried out after obtaining informed consent from the patient.

Lab Investigation:
Lab investigation was advised and the reports were clear.

Radiological Invesgations:
IOPA revealed mild marginal bone loss in maxillary and mandibular anteriors.

Histopathological Examination:
After disinfecting the site with povidone iodine excisional biopsy was done with No. 15 BP blade in relation to 11. Histopathology of the excised lesion exhibits a hyperplastic parakeratinized stratified squamous epithelium of variable thickness. The underlying tissue shows abundant fibrous connective tissue with wavy collagen fibre bundles and few blood vessels. There is a dense infiltration of plasma cells strikingly around the blood vessels and subepithelial region. Within the plasma cell, aggregates of few Russel bodies are noticed.

Based on clinical radiological and histopathological findings and results of various investigations a diagnosis of plasma cell gingivitis was made.

Management:
The patient was asked to refrain from the use of herbal tooth paste which was found to be the causative agent. Scaling and root planning was done with Gracey curettes in maxillary and mandibular anteriors after obtaining informed consent. Complete oral hygiene instructions were given. Kenocort 0.1% oral paste 5gm was prescribed and patient was advised to apply it on the lesion for 1 month. Regular monitoring of the use of kenocart ointment is carried out. Zincovit tablet was prescribed for 1 month. The patient was advised to use a mouth rinse of 0.2%, 10 mL chlorhexidine twice daily along with the use of an ultrasoft toothbrush and dental floss.

There was a shrinkage of the lesion following phase I therapy and the probing depth was recorded as 2mm. The area was checked for plaque removal and home care compliance for the first month on weekly basis and was followed by routine supportive periodontal therapy at 3 months interval. The adjunctive use of antioxidant was added in conventional periodontal therapy. Satisfactory results were obtained following non-surgical periodontal therapy.

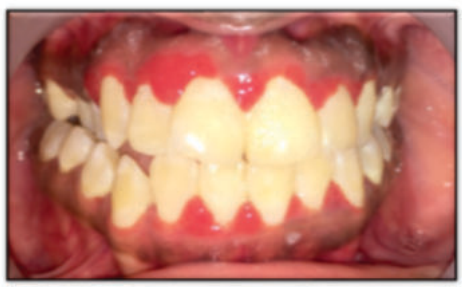
DISCUSSION:
Plasma cell gingivitis is a rare condition, characterized by diffuse and massive infiltration of the plasma cells into the connective tissue. The first case was reported by Kerr et al. in 1981[12], when they observed gingival enlargement in gum chewers, which disappeared following the discontinuation of the chewing habit.

Plasma cell gingivitis was classified by Timms et al into 3 types: caused by an allergen,bearing a neoplastic nature and gingivitis of unknown origin.The etiology in our case was allergy to the use of herbal toothpaste and hence, can be classified as the first variant of PCG. Plasma cell gingivostomatitis is a similar condition, characterized by a triad of cheilitis, glossitis [13], and gingivitis. Plasma cell granuloma, also known as an inflammatory pseudotumor, inflammatory myofibroblastic tumor or xanthomatous pseudotumor is an uncommon non-neoplastic lesion used to describe an enlargement confined to one specific site. The most common location of occurrence is the lungs, but it may also be found in the brain, kidney, heart, and rarely in the gingiva. In our case, the involvement was limited to the gingiva of the anterior segment of the upper and lower jaw.

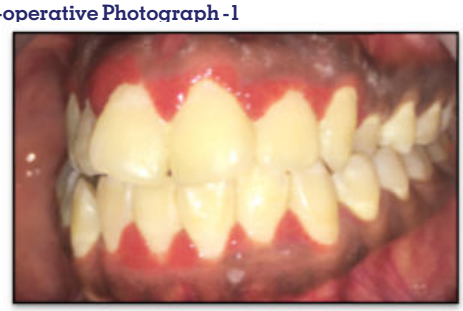
Histologically, this condition must be differentiated from other aggressive conditions, such as, leukemia, plasmacytoma, multiple myeloma, and Waldenstrom's macroglobulinemia, to facilitate early treatment for a better prognosis. Unlike plasmacytomas, the basic proliferative process associated with the growth of plasma cell gingivitis is reactive plasmacytic proliferation. A negative Nikolsky's sign ruled out pemphigus, other cutaneous diseases like pemphigoid, lichen planus and discoid lupus erythematosus. It differs from plaque induced gingivitis because the lesion is in the oral aspect of attached gingiva.

CONCLUSION:
A thorough clinical history usually discloses some of the gingival disturbances. An early diagnosis would direct the clinician toward an appropriate treatment plan, especially for unusual conditions masquerading as common lesions, which are refractory to conventional therapy. Challenging of the offending lesion results in recurrence of the lesion. Regular follow-up and new treatment strategies are therefore required.

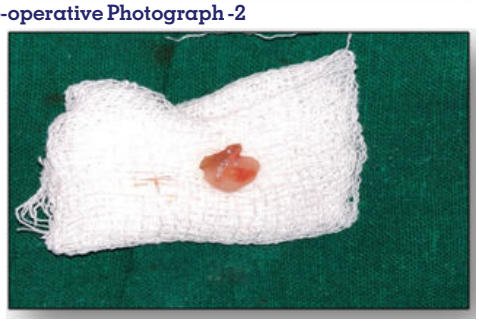
Figures:



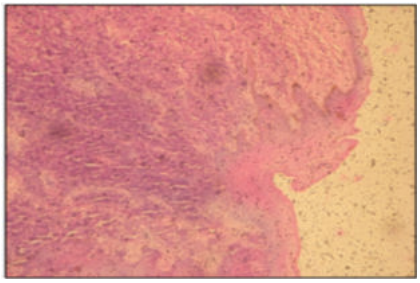
1) Pre-operative Photograph -1



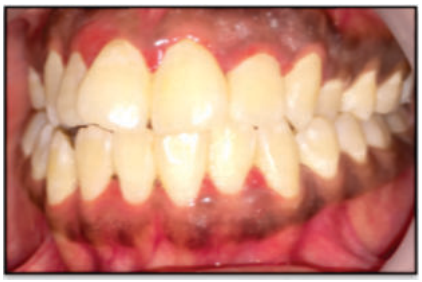
2) Pre-operative Photograph -2



3) Biopsy Specimen



4) Histopathology Photograph



5) Post-operative Photograph

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